EXHIBIT #55C

TABLE 1. Sociodemographic Variations in Waking and Nocturnal ABP in Millimeters of Mercury (mm Hg)

Sociodemographic Variables	Waking SBP $N = 357$	Nocturnal SBP $N = 245$	Waking DBP	Nocturnal DBP
Gender				
Women	129.09 (1.09)*	122.73 (1.72)*	78.27 (0.74)	71.18 (1.15)
Men	137.47 (1.23)	126.75 (1.95)	82.70 (0.84)	73.04 (1.30)
Race/ethnicity				
Black	133.88 (1.07)	125.46 (1.65)	81.48 (0.71)**	73.78 (1.09)**
Latino	130.97 (1.37)	122.97 (2.08)	78.16 (0.91)	69.18 (1.37)
Education level				
<HS	135.91 (1.62)	125.52 (2.55)	81.45 (1.08)	72.62 (1.62)
HS	132.28 (1.08)	125.01 (1.74)	80.14 (0.72)	71.89 (1.11)
College+	130.25 (2.08)	120.77 (3.18)	77.71 (1.39)	68.96 (2.03)
Employment				
Not working	134.71 (1.19)***	127.26 (1.84)***	81.58 (0.79)***	73.20 (1.17)
Working	131.32 (1.16)	121.58 (1.89)	78.79 (0.77)	69.98 (1.20)
Poverty group				
≤1× poverty level	135.07 (1.30)	126.58 (2.04)	81.42 (0.87)	73.15 (1.30)
≤2× poverty level	131.29 (1.61)	124.19 (2.56)	80.14 (1.08)	72.25 (1.63)
≤3× poverty level	133.31 (2.26)	122.00 (3.72)	79.85 (1.52)	69.97 (2.37)
>3× poverty level	130.80 (1.66)	122.57(2.66)	78.08 (1.11)	69.25 (1.70)

Analyses of the effects of education, employment status, and poverty group include BMI, age, gender and race as covariates. The means reported are adjusted for these covariates, with the standard error reported in parentheses.

* Men versus women, $p < .05$.

** Blacks versus Latino(a)s, $p < .01$.

*** Employed versus unemployed, $p < .05$.

Comparisons Between Those With and Without Sleep Data

A larger sample ($n = 357$) was available for analyses of the effects of racism on daytime ABP than that was available for analyses of the effects of racism on nocturnal ABP ($n = 245$). There were no significant differences in hypertensive status between those who did and did not have nocturnal BP data ($\chi^2(3) = 3.89, p > .27$). There were no significant differences between those who did versus did not have nocturnal data on race, gender, age, marital status, education, or employment status (all p values ns). Those with sleep data tended to have slightly higher incomes ($p < .07$). Those with sleep data had significantly lower BMI (mean = 28.00) than those without sleep data (mean BMI = 29.41; $F(1,355) = 5.60, p < .02$).

Demographic Variations in ABP

These analyses examine sociodemographic differences in perceived racism and in waking and nocturnal ABP to determine which variables should be considered as covariates in the main analyses. ANOVAs revealed that there were no main effects of gender, education, occupational status, or income level differences in PEDQ-CV total scores. There were marginal race/ethnicity differences, with Blacks reporting slightly higher levels of exposure than Latino(as) ($F(1,355) = 3.61$, $p < .08$).[2]

Table 1 displays sociodemographic differences in mean waking and nocturnal ABP. Mixed models analyses with gender as the independent variable indicated that in comparison with women, men had significantly higher waking SBP ($F(1,332) = 26.17, p < .0001$) and waking DBP ($F(1,332) = 15.71, p < .0001$), but did not differ in nocturnal SBP ($p < .13$) or DBP ($p < .29$). Race/ethnicity analyses revealed that in comparison with Latino(a)s, Blacks had significantly higher levels of waking DBP ($F(1,332) = 8.27, p < .01$), marginally higher levels of waking SBP ($F(1,332) = 2.81, p < .10$), and significantly higher nocturnal DBP ($F(1,242) = 6.89, p < .01$). As expected, age was positively associated with waking SBP ($B = 0.25$, $SE = 0.09$, $t = 2.95$, $p < .005$), nocturnal SBP ($B = 0.42$, $SE = 0.15$, $t = 2.71$, $p < .01$), waking DBP ($B = 0.25$, $SE = 0.06$, $t = 4.49$, $p < .0001$), and nocturnal DBP ($B = 0.46$, $SE = 0.08$, $t = 5.58$, $p < .001$). Consequently, age, gender, and race/ethnicity were entered as covariates in subsequent analyses.

Racism and ABP

To evaluate the association of perceived racism to ABP, mixed models analyses were performed with perceived racism (PEDQ-Total), time period (waking versus nocturnal), and their interaction serving as predictor variables. Gender, race/ethnicity, age, and BMI served as between-person covariates. Posture served as a within-person covariate. For SBP, the main effect of perceived racism approached significance ($F(1,341) = 2.72, p < .10$), but both the main effect of time period ($F(1,230) = 16.10, p < .001$) and the interaction of perceived racism by time period ($F(1,7449) = 11.08, p < .001$) were highly significant. For DBP, the effect of perceived racism was not significant ($F(1,341) = 1.37, p < .24$), but the main effect of time period ($F(1,230) = 7.83, p < .01$), and the interaction of perceived racism by time period ($F(1,7449) =$

[2]Previously, we have reported that sociodemographic differences in perceived racism depend on the dimension (subscale) of racism under investigation (56).

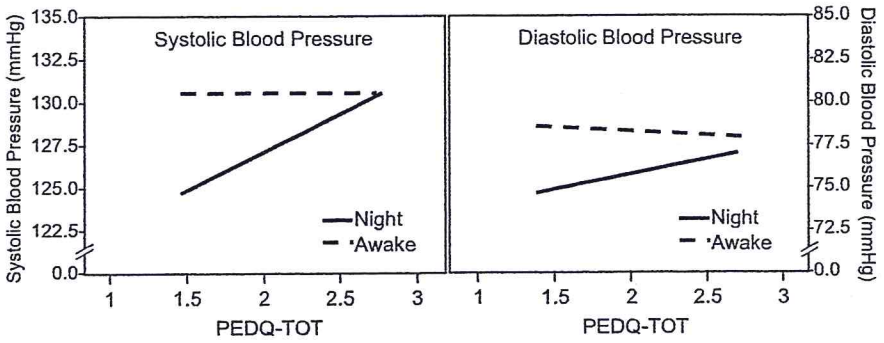

Figure 1. Predicted waking and nocturnal BP as a function of perceived racism (PEDQ-CV lifetime discrimination), adjusted for age, gender, race/ethnicity, body mass index, hostility, and individual SES.

4.41, $p < .04$) were significant. Figure 1 displays predicted scores of the interaction of perceived racism and time period for SBP and DBP, displaying estimated effects of perceived racism on waking and nocturnal ABP. The figures suggest that there is a positive relationship of racism to nocturnal ABP, but not to waking ABP.

Follow-up analyses of the significant interaction effects were conducted to examine the association of perceived racism to ambulatory SBP and DBP separately for waking and nocturnal periods. The results indicate a significant association of perceived racism to nocturnal, but not to waking SBP and DBP. Specifically, the association of perceived racism to waking ambulatory SBP and DBP was not significant ($t < 1$, p values = ns). However, the association of perceived racism to nocturnal SBP ($B = 5.22$, $SE = 1.97$, $df = 238$, $t = 2.64$, $p < .01$) was significant and the association of perceived racism to nocturnal DBP ($B = 2.40$, $SE = 1.26$, $df = 238$, $t = 1.90$, $p < .06$) approached significance.[3]

Both diabetes and morbid obesity have been associated with nocturnal BP (57,58). Therefore, all analyses were controlled for BMI, and we repeated the analyses eliminating individuals with diabetes. The effects of perceived racism on nocturnal ABP remained significant for both SBP and DBP when diabetics ($n = 18$) were removed from the sample.[4]

Subscale Analyses

The primary analyses were repeated with the four subscales of the PEDQ-CV entered instead of the lifetime score to determine if any subscale was uniquely related to ABP, controlling for the effects of the others. For nocturnal DBP, stigmatization was the only scale to emerge as independently associated ($B = 2.63$, $SE = 1.33$, $df = 235$, $t = 1.98$, $p < .04$). For nocturnal SBP, no single scale emerged as a significant predictor controlling for the effects of the other scales.

Effects of Trait Hostility on Perceived Racism

Hostility was positively associated with perceived racism ($r = 0.23$, $p < .001$), and was weakly associated with waking DBP ($p < .10$) but was unrelated to nocturnal BP. With hostility included as an additional covariate the interaction of perceived racism by time period was significant for both SBP ($p < .001$) and DBP ($p < .05$), and the associations of racism to nocturnal SBP ($p < .01$) and DBP ($p < .05$) also remained significant.

Effects of Socioeconomic Status on Perceived Racism

Table 1 displays mean ABP (adjusted for race, gender, and age) for education level and employment status. Poverty ratios were calculated by dividing the participant's gross household income by the poverty level income for households with equivalent numbers of members, seniors citizens, and children (18 and below). A poverty ratio of one indicates that the gross household income is equivalent to the poverty level for a household of similar composition. Participants were divided into income level groups based on the ratio of their gross household income to the poverty level income for households with equivalent numbers of members, senior citizens, and children (18 and below). Four income level groups were constructed: a) Group 1 (≤ poverty level), income at or below poverty level for households of their size and composition; b) Group 2 (≤2× poverty level), income more than the poverty level, but less than twice the poverty level; c) Group 3 (≤3× poverty level), income more than twice the poverty level, but less than three times the poverty level; and d) Group 4 (>3× poverty level), income more than three times the poverty level.

Mixed models analyses controlling for BMI, age, gender, and race revealed marginal effects of education on waking SBP ($p < .08$) and DBP ($p < .11$), and nonsignificant associations with nocturnal BP (p values < 0.30). Employment (versus unemployment) was associated with lower levels of waking SBP ($F(1,329) = 4.19$, $p < .05$), DBP ($F(1,329) = 6.39$, $p < .02$) and nocturnal SBP ($F(1,239) = 4.51$, $p < .04$), and marginally associated with nocturnal DBP ($F(1,239) =$

[3]In the primary analyses all 357 participants are included. However, the main effect and interaction effects are equivalent if the analyses are confined to the 245 individuals who have both waking and nocturnal readings. The effects of perceived racism on waking BP remain nonsignificant when analyses are confined to the subset with sleep readings. The effects of perceived racism on nocturnal SBP remain significant and the effects of perceived racism on DBP achieve significance ($p < .03$) when analyses are confined to the subset of participants with 3 or more nocturnal readings.

[4]In addition, the effects remain significant for SBP and marginally significant for DBP ($p < .13$) when individuals with Stage II or III obesity (i.e., BMI > 35, $n = 42$) were removed from sample.

3.57, $p < .06$). There were no significant relations of poverty group to waking or nocturnal SBP or DBP.

We repeated the primary analyses with the three measures of individual SES and hostility included in the analyses. The interaction of PEDQ and Time Period remained significant for both SBP ($p < .001$) and DBP ($p < .05$). Follow-up analyses performed by time period and with these additional covariates indicated that the effects of perceived racism on nocturnal SBP ($B = 5.16$, $SE = 2.04$, $df = 234$, $t = 2.52$, $p < .02$) remained significant and the effects on DBP approached significance ($B = 2.34$, $SE = 1.31$, $df = 234$, $t = 1.79$, $p < .08$). The effects are significant for both SBP and DBP when the analyses are confined to the 183 individuals with three or more nocturnal readings. Marital status is associated with SES, but the effects of racism on nocturnal BP also remained significant when marital status was included as an additional covariate.

Blood Pressure Dipping

Conventionally, physicians use nocturnal dipping status (i.e., a change from waking to nocturnal BP of 10% or greater) as an index of cardiovascular risk (59). To assess the relationship of perceived racism to nocturnal dipping, we conducted a logistic regression analysis with age, race/ethnicity, gender, and BMI serving as covariates, perceived racism serving as a predictor and dipping status (yes or no) serving as the outcome. The effects of perceived racism were significant (estimate = 0.53, SE = 0.26, $p < .05$). The odds ratio associated with each standard deviation increase in perceived racism was 1.40 (95% CI, 1.01–1.93), suggesting that for each 1 SD increase in PEDQ score, the odds of being a nondipper increased by 40%. When we included only those individuals with three or more nocturnal readings ($n = 183$), the odds ratio increases to 1.70 (95% CI, 1.02–2.84). The effects remain significant and the odds ratio does not change when sociodemographic and personality covariates are included.

DISCUSSION

The aim of the present study was to test the hypothesis that perceived racism was associated with ABP in a sample of community-dwelling adults. Results indicated that perceived racism was associated with nocturnal SBP and nocturnal DBP, but not with daytime ABP. Additionally, perceived racism substantially increases the risk of failing to display the expected pattern of nocturnal blood pressure dipping.

The findings further suggest that the relationship of racism to nocturnal ABP is not a function of trait hostility, specifically the tendency to attribute hostile intent to others or to view the world in a cynical manner. In addition, employment status and poverty ratio were associated with ABP in this sample. However, the effects of racism on nocturnal ABP persisted despite controlling for these variables and for education level. This suggests that racism is a unique psychosocial stressor, exerting effects on cardiovascular system above and beyond traditional stressors such as low SES and hostility.

These results are partly consistent with those of Steffen et al. (33) who reported an association of perceived racism to daytime but not nocturnal ABP. It is possible that we did not find an association between racism and daytime ABP (even in the larger sample with available daytime readings), because our sample included both employed and unemployed individuals whose activities and life demands may be more diverse than those of the individuals included in the sample of employed individuals included in the study by Steffen et al. (33). Activities during the day may have been too varied, both in emotional and physical demands, to easily evaluate the effects of a single individual difference variable (i.e., racism) on daytime cardiovascular response. Gerin et al. (60) have found, for example, that a distraction tends to attenuate angry thoughts, as well as BP elevation, in the laboratory; and the natural environment comprises many distractions that may curtail rumination. Regional or sample-specific variations in the association of racism to daily stress or coping may also account for differences in the findings.

As investigators have suggested, the association of perceived racism to BP may depend in part on the link between exposure to maltreatment and the strategies for coping with maltreatment (19). For example, in the same sample in which racism was associated with day-time ABP, Steffen et al. (33) reported that anger suppression is associated with nocturnal ABP. In prior studies, we have shown that perceived racism was positively related to both experiences of anger and anger suppression (10,41). Future analyses will examine the role of anger and anger suppression on the relationship of racism to ABP.

In contrast, the effects of racism may be more discernible at night without the events of the day available to distract the individual. As we have demonstrated previously, perceived racism seems to be positively associated with the intensity of daily negative social interactions and daily negative mood, as well as threat appraisals (41). These daily stressors and distress responses may be associated with more difficulty in reducing psychophysiological activation at night. Individuals may remain in a persistent state of distress, even if they do not attribute the cause of their distress to racism, or they may ruminate about specific episodes of unfair treatment. Both distress and rumination may disrupt sleep or contribute to heightened arousal during sleep (61,62).

LIMITATIONS

The nocturnal BP data do not represent a true measure of sleep BP, because we did not obtain measures of movement or polysomnography. Instead, we used the participant's self-report of the time they went to sleep. It is possible that perceived racism is actually associated with sleep quality and that the higher BP levels observed in those with high levels of racism reflect sleep difficulties (i.e., the participant is lying down and resting but unable to sleep during these periods (63)). New research suggests that American Blacks report difficulties with sleep and that perceived racism is associated with sleep quality (63,64). Further research with controls for

subjective and objective indices of sleep quality will be necessary to determine if the effects of racism on nocturnal BP are a function of sleep difficulties or of stress-related effects on autonomic control during sleep.

Both diabetes and morbid obesity are associated with elevated nocturnal BP. However, in addition to controlling for these variables, we also reanalyzed the data with individuals who were diabetic or morbidly obese removed from the sample. The effects of racism on nocturnal BP remained significant with these individuals eliminated from the analyses.

Only 69% of the sample completed the nocturnal monitoring. These individuals had lower BMI scores than those who did not complete the sleep study, but did not differ from those who did not complete nocturnal monitoring on reports of racism, personality measures, or SES. Finally, the current sample is under-powered to appropriately test race/ethnicity differences in the association between racism and BP. Future analyses will examine ethnicity effects.

SUMMARY AND CONCLUSIONS

Overall, the data support the hypothesis that perceived racism is associated with nocturnal BP and an increased risk of nondipping in a population of community-dwelling urban Black and Hispanic adults. Further, the findings indicate that the observed effects are not a function of poverty or of a general tendency to view the world as unfair or threatening. Given the link between nocturnal BP and cardiovascular morbidity, these results suggest a mechanism that may potentially explain racial disparities in HTN.

The authors thank many research assistants who were critical to the success of this project: Jenny Atencio, Karina Bienfait, Nisha Brady, Mara Faust, Juhee Jhalani, Jasmine Kwok, Asha Kumar, Harrald Magny, Aida Mencia-Ripley, and Jahanara Ullah.

REFERENCES

1. National Institute of Health. The Health of the Nation: Highlights of the Healthy People 2000 Goals. 1995 Report on Progress [On-line]. 2000. Available at: http://www.healthypeople.gov/Data/2010prog/focus12/2007Focus12.pdf. Accessed December 3, 2007.
2. Anderson NB, McNeilly M, Myers H. Toward understanding race difference in autonomic reactivity: a proposed contextual model. In: Turner JR, Sherwood A, Light KC, editors. Individual differences in cardiovascular response to stress perspectives on individual differences. New York: Plenum Press; 1992:125–45.
3. Brondolo E, Rieppi R, Kelly KP, Gerin W. Perceived racism and blood pressure: a review of the literature and conceptual and methodological critique. Ann Behav Med 2003;25:55–65.
4. Clark R. Perceptions of interethnic group racism predict increased vascular reactivity to a laboratory challenge in college women. Ann Behav Med 2000;22:214–22.
5. Clark R, Anderson NB, Clark VR, Williams DR. Racism as a stressor for African Americans. Am Psychol 1999;54:805–16.
6. Contrada RJ, Ashmore RD, Gary ML, Coups E, Egeth JD, Sewell A, Ewell K, Goyal TM, Chasse V. Ethnicity-related sources of stress and their effects on well-being. Curr Dir Psychol Sci 2000;9:136–9.
7. Contrada RJ, Ashmore RD, Gary ML, Coups E, Egeth JD, Sewell A, Ewell K, Goyal T. Measures of ethnicity-related stress: psychometric properties, ethnic group differences, and associations with well-being. J Appl Soc Psychol 2001;31:1775–820.
8. Brondolo E, Karlin W, Alexander K, Bobrow A, Schwartz J. Workday communication and ambulatory blood pressure: implications for the reactivity hypothesis. Psychophysiology 1999;36:86–94.
9. Suls J, Wan CK. The relationship between trait hostility and cardiovascular reactivity: a quantitative review and analysis. Psychophysiology 1993;30:615–26.
10. Brondolo E, Thompson S, Brady N, Appel R, Cassells A, Tobin JN, Sweeney M. The relationship of racism to appraisals and coping in a community sample. Ethn Dis 2006;15:14–19.
11. Harrell JP, Harrell JP, Hall S, Taliaferro J. Physiological responses to racism and discrimination: an assessment of the evidence. Am J Public Health 2003;93:243–8.
12. Paradies Y. A systematic review of empirical research on self-reported racism and health. Int J Epidemiol 2006;35:888–901.
13. Broman CL. The health consequences of racial discrimination: a study of African Americans. Ethn Dis 1996;6:148–53.
14. Clark R, Gochett P. Interactive effects of perceived racism and coping responses predict a school-based assessment of blood pressure in black youth. Ann Behav Med 2006;32:1–9.
15. Dressler WW. Social identity and arterial blood pressure in the African-American community. Ethn Dis 1996;6:176–89.
16. James SA, LaCroix AZ, Kleinbaum DG, Strogatz DS. John Henryism and blood pressure differences among Black men. II. The role of occupational stressors. J Behav Med 1984;7:259–75.
17. James K, Lovato C, Khoo G. Social identity correlates of minority workers' health. Acad Manage J 1994;37:383–96.
18. Krieger N. Racial and gender discrimination: risk factors for high blood pressure? Soc Sci Med 1990;30:1273–81.
19. Krieger N, Sidney S. Racial discrimination and blood pressure: the CARDIA study of young Black and White adults. Am J Public Health 1996;86:1370–8.
20. Peters RM. Racism and hypertension among African Americans. West J Nurs Res 2004;26:612–31.
21. Armstead CA, Lawler KA, Gorden G, Cross J, Gibbons J. Relationship of racial stressors to blood pressure responses and anger expression in Black college students. Health Psychol 1989;8:541–56.
22. Clark R. Self-reported racism and social support predict blood pressure reactivity in Blacks. Ann Behav Med 2003;25:127–36.
23. Clark R. Perceived racism and vascular reactivity in Black college women: moderating effects of seeking social support. Health Psychol 2006;25:20–5.
24. Clark R, Adams JH. Moderating effects of perceived racism on John Henryism and blood pressure reactivity in Black female college students. Ann Behav Med 2004;28:126–31.
25. Fang CY, Myers HF. The effects of racial stressors and hostility on cardiovascular reactivity in African American and Caucasian men. Health Psychol 2001;20:64–70.
26. Guyll M, Matthews KA, Bromberger JT. Discrimination and unfair treatment: relationship to cardiovascular reactivity among African American and European American women. Health Psychol 2001;20:315–25.
27. Merritt MM, Bennett GG Jr, Williams RB, Edwards CL, Sollers JJ III. Perceived racism and cardiovascular reactivity and recovery to personally relevant stress. Health Psychol 2006;25:364–9.
28. Staessen JA, Thijs L, Fagard R, O'Brien ET, Clement D, de Leeuw PW, Mancia G, Nachev C, Palatini P, Parati G, Tuomilehto J, Webster J. Predicting cardiovascular risk using conventional vs. ambulatory blood pressure in older patients with systolic hypertension. Systolic Hypertension in Europe Trial Investigators.[see comment] JAMA 1999;282:539–46.
29. Pickering TG, Shimbo D, Haas D. Ambulatory blood-pressure monitoring. N Engl J Med 2006;354:2368–74.
30. Ohkubo T, Imai Y, Tsuji I, Nagai K, Watanabe N, Minami N, Kato J, Kikuchi N, Nishiyama A, Aihara A, Sekino M, Satoh H, Hisamichi S. Relation between nocturnal decline in blood pressure and mortality. The Ohasama Study. Am J Hypertens 1997;10:1201–7.
31. Ohkubo T, Hozawa A, Yamaguchi J, Kikuya M, Ohmori K, Michimata M, Matsubara M, Hashimoto J, Hoshi H, Araki T, Tsuji I, Satoh H, Hisamichi S, Imai Y. Prognostic significance of the nocturnal decline in blood pressure in individuals with and without high 24-h blood pressure: the Ohasama study. [see comment]. J Hypertens 2002;20:2183–9.
32. Matthews KA, Salomon K, Kenyon K, Zhou F. Unfair treatment, discrimination, and ambulatory blood pressure in black and white adolescents. Health Psychol 2005;24:258–65.
33. Steffen PR, McNeilly M, Anderson N, Sherwood A. Effects of perceived racism and anger inhibition on ambulatory blood pressure in African Americans. Psychosom Med 2003;65:746–50.
34. Profant J, Dimsdale JE. Psychosocial factors and congestive heart failure. Int J Behav Med 2000;7:236–55.

35. Fumo MT, Teeger S, Lang RM, Bednarz J, Sareli P, Murphy MB. Diurnal blood pressure variation and cardiac mass in American blacks and whites and South African blacks. Am J Hypertens 1992;5:111–16.
36. Wilson DK, Kliewer W, Teasley N, Plybon L, Sica DA. Violence exposure, catecholamine excretion, and blood pressure nondipping status in African American male versus female adolescents. Psychosom Med 2002;64:906–15.
37. Bishop GD, Pek J, Ngau F. Ethnicity, sex, trait anger, and nocturnal blood pressure decline. Psychophysiology 2005;42:290–7.
38. Ituarte PH, Kamarck TW, Thompson HS, Bacanu S. Psychosocial mediators of racial differences in nighttime blood pressure dipping among normotensive adults. Health Psychol 1999;18:393–402.
39. Rau R, Triemer A. Overtime in relation to blood pressure and mood during work, leisure, and night time. Soc Indic Res 2004;67:51–73.
40. Thomas KS, Nelesen RA, Dimsdale JE. Relationships between hostility, anger expression, and blood pressure dipping in an ethnically diverse sample. Psychosom Med 2004;66:298–304.
41. Broudy R, Brondolo E, Coakley V, Brady N, Cassells A, Tobin J, Sweeney M. Perceived ethnic discrimination in relation to daily moods and negative social interactions. J Behav Med 2007;30:31–43.
42. Brondolo E, Kelly KP, Coakley V, Gordon T, Thompson S, Levy E, Gordon A, Tobin JN, Sweeney M, Contrada R. The perceived ethnic discrimination questionnaire: development and preliminary validation of a community version. J Appl Soc Psychol 2005;35:335–65.
43. Brondolo E, Rieppi R, Erickson SA, Bagiella E, Shapiro PA, McKinley P, Sloan R. Hostility, interpersonal interactions, and ambulatory blood pressure. Psychosom Med 2003;65:1003–11.
44. Kessler RC, Mickelson KD, Williams DR. The prevalence, distribution, and mental health correlates of perceived discrimination in the United States. J Health Soc Behav 1999;40:208–30.
45. Williams D. Race, socioeconomic status, and health: the added effects of racism and discrimination. In: Adler NE, Marmot M, McEwen BS, Stewart J, editors. Socioeconomic status and health in industrialized nations: social, psychological, and biological pathways. Vol 896. New York: New York Academy of Sciences; 1999:173–88.
46. Pickering T. Cardiovascular pathways: socioeconomic status and stress effects on hypertension and cardiovascular function. Ann N Y Acad Sci 1999;896:262–77.
47. Perloff D, Grim C, Flack J, Frohlich ED, Hill M, McDonald M, Morgenstern BZ. Human blood pressure determination by sphygmomanometry. Circulation 1993;88:2460–70.
48. Barefoot JC, Dodge KA, Peterson BL, Dahlstrom WG. The Cook-Medley hostility scale: item content and ability to predict survival. Psychosom Med 1989;51:46–57.
49. Karlin W, Brondolo E, Schwartz J. Workplace social support and ambulatory cardiovascular activity in New York City traffic agents. Psychosom Med 2003;65:167–76.
50. White WB, Morganroth J. Usefulness of ambulatory monitoring of blood pressure in assessing antihypertensive therapy. Am J Cardiol 1989;63: 94–8.
51. Littell RC, Milliken GA, Stroup WWW, Wolfinger RD. SAS system for mixed models. Cary, NC: SAS Institute; 1996.
52. Schwartz J, Stone A. Strategies for analyzing ecological momentary assessment data. Health Psychol 1998;17:6–16.
53. Wolfinger RD, Chang M. Comparing SAS GLM and MIXED procedures for repeated measures. SAS user's group international conference proceeding. SUGI 1995;20:1172–82.
54. Jamner LD, Shapiro K, Goldstein IB, Hug R. Ambulatory blood pressure and heart rate in paramedics: effects of cynical hostility and defensiveness. Psychosom Med 1991;53:393–406.
55. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 Report. JAMA 2003;289:2560–72.
56. Brondolo E, Beatty D, Cubbin C, Weinstein M, Saegert S, Wellington RL, Tobin J, Cassells A, Schwartz JE. Sociodemographic variations in self-reported racism in a community sample of Blacks and Latino(a)s. J Appl Soc Psychol. In press.
57. Nakano S, Ito T, Furuya K, Tsuda S, Konishi K, Nishizawa M, Nakagawa A, Kigoshi T, Uchida K. Ambulatory blood pressure level rather than dipper/nondipper status predicts vascular events in type 2 diabetic subjects. Hypertens Res 2004;27:647–56.
58. Kotsis V, Stabouli S, Bouldin M, Low A, Toumanidis S, Zakopoulos N. Impact of obesity on 24-hour ambulatory blood pressure and hypertension. Hypertension 2005;45:602–7.
59. Pickering TG. The clinical significance of diurnal blood pressure variations: dippers and nondippers. Circulation 1990;81:700–2.
60. Gerin W, Davidson KW, Christenfeld NJS, Goyal T, Schwartz JE. The role of angry rumination and distraction in blood pressure recovery from emotional arousal. Psychosom Med 2006;68:64–72.
61. Thomsen DK, Yung Mehlsen M, Christensen S, Zachariae R. Rumination–relationship with negative mood and sleep quality. Pers Individ Diff 2003;34:1293–301.
62. Backhaus J, Junghanns K, Hohagen F. Sleep disturbances are correlated with decreased morning awakening salivary cortisol. Psychoneuroendocrinology 2004;29:1184–91.
63. Steffen PR, Bowden M. Sleep disturbance mediates the relationship between perceived racism and depressive symptoms. Ethn Dis 2006;16: 16–21.
64. Dimsdale JE, Hall M, Matthews KA, Okun ML, Irwin MR. Nocturnal reverberations of adversity: the effect of social class, ethnicity, and stress on nocturnal blood pressure and sleep architecture. Denver, CO: American Psychosomatic Society; 2006.

Authorization to Discuss Medical Records or Findings/Observations

I Larry A. Boss do hereby grant permission to Dr. Robert Fajardo to discuss my medical records or findings/observations with Dr. Daniel Sisbarro and Dr. Matthew McCord of Loyola University Health System.

LUHS: (708) 327-7000

Signature: Larry A. Boss Date: 2.11.2010

Dr. Sisbarro (708) 216-8757 dsisbarr@lumc.edu
Dr. McCord (708) 216-6755
P+S (708) 216-4702 22 (Ms Coyd sp.)
FAX# (708) 216-5617

From: "Larry A. Boss" <laboss99@comcast.net>
To: "Larry A. Boss" <laboss99@comcast.net>
Sent: Wednesday, February 10, 2010 7:00 PM
Subject: Fw: Permission to Discuss my Medical History

Dr. Daniel Sisbarro 708/216-8757

----- Original Message -----
From: "Larry A. Boss" <laboss99@comcast.net>
To: <larry_a._boss@hud.gov>
Sent: Wednesday, February 10, 2010 6:28 PM
Subject: Fw: Permission to Discuss my Medical History

>
> ----- Original Message -----
> From: "Daniel Sisbarro" <dsisbar@lumc.edu>
> To: "Larry A. Boss" <laboss99@comcast.net>
> Sent: Wednesday, February 10, 2010 5:58 PM
> Subject: Re: Permission to Discuss my Medical History
>
>
> As long as it is ok with you, I am happy to discuss with him and hear his
> thoughts.
>
> dan sisbarro
>
>>>> "Larry A. Boss" <laboss99@comcast.net> 2/10/2010 5:38 PM >>>
> Dr. Sisbarro,
>
> I'm seeing a Dr.Robert Fajardo, (a psychiatrist - job stress) he has been
> reviewing my medical records and has some interesting observations
> regarding the dizzy episodes I was experiencing last summer. Since you are
> my primary care phyisician, he said he would like to talk with you.
>
> I gave him my permission to do so - is this OK with you?
>
> Thanks,
>
> Larry A. Boss
>
>
>

Regular Article

Psychotherapy and Psychosomatics

Psychother Psychosom 2008;77:372–376
DOI: 10.1159/000151517

Published online: August 21, 2008

Minor Psychiatric Disorders and Syncope: The Role of Psychopathology in the Expression of Vasovagal Reflex

Dionyssios Leftheriotis[a] Ioannis Michopoulos[b] Panayota Flevari[a]
Athanassios Douzenis[b] Christoforos Koborozos[a] Anna Kostopoulou[c]
George N. Theodorakis[c] Lefteris Lykouras[b] Dimitrios T. Kremastinos[a]

[a]Department of Cardiology and [b]Second Department of Psychiatry, 'Attikon' University Hospital of Athens, and [c]Second Department of Cardiology, Onassis Cardiac Surgery Center, Athens, Greece

Key Words
Minor psychiatric disorders · Vasovagal syncope · Tilt testing · Somatization

Abstract
Background: A high prevalence of minor psychiatric disorders (MPDs) has been reported in patients with vasovagal syncope (VVS). However, the relationship between the psychiatric substrate and syncope remains unclear. ***Methods:*** In order to test the hypothesis that MPDs may predispose to VVS, we assessed the prevalence of syncope, the response to head-up tilt test (HUTT) and the efficacy of psychiatric drug treatment in reducing syncopal episodes, in patients with recently diagnosed MPDs. The response to HUTT was compared with that in an equal number of matched (a) patients with VVS and (b) healthy controls. ***Results:*** A high rate of patients with MPDs (58%) had a positive HUTT. Additionally, 45% had a history of syncope; among them, the rate of positive HUTT was identical to that in the VVS group (83%). Following psychiatric drug treatment, the number of patients with syncope decreased in the MPD group (6/67 from 30/67, $p < 0.01$). Psychiatric symptoms and quality of life were also improved. The number of syncopal spells decreased equally in the MPD and VVS groups (0.6 ± 0.5 from 2.5 ± 1.4, $p < 0.01$, and 0.7 ± 0.5 from 2.7 ± 1.3, $p < 0.01$, respectively). ***Conclusion:*** A high proportion of patients with MPDs experience syncope, associated with a high rate of positive HUTT, comparable to that observed in VVS. Psychiatric treatment results in the improvement of syncopal and psychiatric symptoms. These findings suggest involvement of co-occurring MPDs in the pathogenesis of VVS. Therefore, the diagnosis and treatment of MPDs, when present, may be crucial for the effective therapy of vasovagal syndrome.

Vasovagal syncope (VVS) is the most common cause of syncope in the general population. It is characterized by a sudden failure of the autonomic nervous system to maintain blood pressure and heart rate at a level sufficient to obtain cerebral perfusion and consciousness [1]. Recurrent episodes of VVS can result in injury and may provoke substantial anxiety and a degree of functional impairment among patients [2, 3]. However, the treatment of VVS still remains a therapeutic challenge.

A high prevalence of 'minor psychiatric disorders' (MPDs) has been reported among patients with vasovagal syndrome [4]. Although there are indications that central serotonergic activity participates in the pathogen-

KARGER

Fax +41 61 306 12 34
E-Mail karger@karger.ch
www.karger.com

0033–3190/08/0776–0372$24.50/0

Accessible online at:
www.karger.com/pps

Dionyssios Leftheriotis, MD
'Attikon' University Hospital of Athens
1 Rimini Street, Haidari
GR–124 62 Athens (Greece)
Tel. +30 210 583 2355, Fax +30 210 583 1351, E-Mail dionle1@otenet.gr